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Issue Date: 04 September 2007

CASE NO.: 2006-LHC-00698

OWCP NO.: 01-147272

In the Matter of:

J. S..[1]
Claimant

v.

ELECTRIC BOAT CORPORATION
Employer / Self-Insurer

Appearances:

Stephen C. Embry (Embry & Neusner),
Groton, Connecticut, for the Claimant

Edward W. Murphy (Morrison Mahoney),
Boston, Massachusetts, for the Employer

Before: Daniel F. Sutton
Administrative Law Judge

DECISION AND ORDER AWARDINGING BENEFITS

I. Statement of the Case

A. S. (the "Decedent"), who worked for Electric Boat Corporation ("Employer" or "EB") from 1963 until he retired in 1998, was awarded workers' compensation benefits in 2000 under the Longshore and Harbor Workers' Compensation Act, as amended, 33 USC § 901, *et seq.* ("the Act") by ALJ David W. Di Nardi based on a finding that he had sustained a permanent partial lung impairment caused by his exposure to and inhalation of asbestos dust and fibers and other injurious pulmonary stimuli while working at EB. He was subsequently diagnosed with cancer and died on January 6, 2005. The Decedent's wife, J. S. (the "Claimant"), now brings this claim, alleging that his death was caused by exposure to asbestos and other carcinogens during the

[1] [1] In accordance with Claimant Name Policy which became effective on August 1, 2006, the Office of Administrative Law Judges uses a claimant's initials in published decisions in lieu of the claimant's full name. See Chief ALJ Memorandum dated July 3, 2006 available at http://www.oalj.dol.gov/PUBLIC/RULES_OF_PRACTICE/REFERENCES/MISCELLANEOUS/CLAIMANT_NAME_POLICY_PUBLIC_ANNOUNCEMENT.PDF.

course of his employment at EB. After an informal hearing before the District Director of the Department of Labor's Office of Workers' Compensation Programs ("OWCP"), the claim was transferred to the Office of Administrative Law Judges ("OALJ") for formal hearing pursuant to section 19(d) of the Act. 33 USC § 919(d).

On June 12, 2006 a formal hearing was conducted in New London, Connecticut. Both parties were represented by counsel. The Claimant testified, and she called two additional witnesses. Documentary evidence was admitted as Claimant's Exhibits ("CX") 1-9, and 11, and Employer's Exhibits ("EX") 1, 2, and 4-9. Procedural documents were admitted as ALJ Exhibits ("ALJX") 1-16, and stipulations were introduced as Joint Exhibit ("JX") 1. Hearing Transcript ("TR") 6-22. By order issued on July 6, 2006, a redacted version of Employer's Exhibit 3 was entered into evidence, as well as Claimant's Exhibits 10 and 12. Finally, by an order dated August 4, 2006, the deposition of Milo Pulde, M.D. was admitted as Employer's Exhibit 11. The record was closed on August 4, 2006, and the parties filed helpful post-hearing briefs.

After consideration of the evidence, and the parties' arguments, I conclude that the Claimant has met her burden of proving that the Decedent's death was caused by an occupational disease arising out of and in the course of his employment with Electric Boat. Accordingly, I conclude that she is entitled to death benefits under the Act.

II. The Claim, Stipulations and Issues Presented

In her pre-hearing statement and at the hearing, the Claimant stated that she is seeking an award of death benefits consisting of survivors compensation from January 6, 2005 based on an average weekly wage of $822.40 and funeral and medical expenses. ALJX 6; TR 11. In her post-hearing brief, the Claimant assets an additional claim on behalf of the Decedent's estate for approximately one week of permanent total disability compensation from December 29, 2004 to January 6, 2005. Claimant Br. at 19.

At the hearing, the parties stipulated that: (1) the Act applies to the claim; (2) the diagnosis was made on December 29, 2004, and death was on January 6, 2005; (3) the injury occurred at Groton, Connecticut; (4) there was an employer/employee relationship at all relevant times; (5) the Employer was timely notified of the injury; (6) the claim for benefits was timely filed; (7) the notice of controversion was timely filed; (8) the informal conference was held on January 11, 2006; and, (9) benefits were paid from August 5, 1999 until the date of death as per Administrative Law Judge Di Nardi's decision and order. JX 1.

The issues presented for adjudication are (1) whether the Decedent's death was caused by his employment at EB and (2) if benefits are awarded whether they should be based at the hearing were those of causation of the injury and average weekly wage. JX 1; TR 11-12. In addition, the Claimant's post-hearing assertion of the claim for additional disability compensation raises a due process issue since EB had no prior notice and opportunity to offer responsive evidence and argument.

III. Summary of the Evidence

A. Occupational History

By the time the instant claim was filed, the Decedent had died. However, he was deposed for the earlier proceeding, and the transcript of that deposition was entered into evidence as Claimant's Exhibit 9. The Decedent testified at the deposition that he dropped out of school after the ninth grade and worked as a delivery man for a furniture store. CX-9 at 5. After that, he worked with a company doing surveying for the construction of Interstate 95. *Id.* at 6. He was married in 1961 and subsequently took a job in a plastics plant. *Id.* at 7. To the best of his knowledge, he was not exposed to any asbestos while he worked in the plastics plant. *Id.* at 8. The plant closed down, and the Decedent was hired by EB in January of 1963 as a painter/cleaner in EB's Groton, Connecticut shipyard. *Id.* at 8-9. He described his job as a painter/cleaner as follows: "You painted the boats, and you cleaned the boats. Sometimes you'd be cleaning, sometimes you'd be painting." *Id.* at 9. He worked on both new vessel construction and overhauls of older vessels. *Id.* He testified that he sometimes worked along side "laggers" and that he would clean up asbestos insulation that the laggers removed from pipes. *Id.* at 10. The Decedent said that his only dust protection was a rag tied around his face, but in later years, employees were provided paper masks, and, eventually, respirators. *Id.* at 10-11. He also testified that he extensively used paint thinners and various paints that are known today to be dangerous. *Id.* at 12. He worked in the painter/cleaner classification at EB until December 31, 1998 when he accepted an early retirement package. ALJX 15 (ALJ Di Nardi Decision and Order) at 3.

The Claimant called two of the Decedent's former co-workers at EB to testify at the hearing about the Decedent's working conditions and exposures at EB.[2] The first witness, E.T., began working at EB in 1958 as a painter in Groton, and he retired in 1991. TR 38-39. ET testified that he and the Decedent lived in the same neighborhood before Decedent began working for EB, and they worked together at EB from 1963 to the mid-1970s. *Id.* at 39-41. He said that they initially worked on reconditioning old ships. *Id.* They ripped out piping and generators and worked weekends to clean the vessels so construction could resume on Monday. *Id.* E.T. testified that he and the Decedent cleaned asbestos from decks after it had been ripped off of piping by laggers. TR 44. He stated that they picked up the larger pieces of asbestos and swept up the dust and smaller pieces. *Id.* at 46. Mr. Taylor testified that he could see pieces of asbestos floating in the air and that that there was so much asbestos in the air and on their clothing that it appeared "like sparkles or something on you." *Id.* at 44-45. He said that that they would fill as many as 20-25 five gallon plastic buckets with asbestos on a given day. *Id.* He also described blowing asbestos dust out of the ship's bilges which produced an atmosphere resembling a "blizzard." *Id.* at 48.

E. T. further testified he and the Decedent also performed sand blasting, and applied Tarcet, Devron, Seravon and chromate paints to ships. TR 48-49. He testified that the sandblasting generated a great deal of dust, though they did use respirators occasionally. *Id.* at 49-50. According to E.T., Devron was toxic, use of Seravon was terminated because of its

[2] The Claimant testified at the hearing, but she provided no information on the Decedent's work history at EB.

danger, and chromate paints were also very dangerous. *Id.* at 51-52. He also stated that they worked in close proximity to grinders and welders and could smell the fumes produced by welding. *Id.* at 52-53. He stated that EB workers became aware of the danger of exposure to dust and fumes around 1971 or 1972 after a survey was conducted by a team from the Mt. Sinai Hospital in New York. *Id.* at 53. From that time forward, EB notified workers of the dangers and began providing them with safety equipment. *Id.* at 54. E. T. said that prior to the introduction of breathing protection at EB, workers would tie rags around their faces for dust protection. *Id.* at 64. As a result of the examinations that he underwent in connection with the occupational health surveys at EB in the early 1970s, E. T. only performed minimal work aboard boats after the mid-1970s. *Id.* at 55-56.

H. G., the second former co-worker called at the hearing by the Claimant, was employed as a painter by EB from 1965 until 1996. TR 69. He testified that he and Decedent worked together everyday for four or five years on various projects aboard the ships. *Id.* at 70-71. He and the Decedent cleaned up asbestos insulation after it had been removed by laggers, using "brooms for the big stuff, and the vacuum cleaner for, you know, small debris." *Id.* at 71-72. He testified that as a result of the cleaning work, asbestos dust filled the air and that he would expel asbestos dust when he blew his nose at the end of a shift. *Id.* at 72-74. H. G. also testified that he and the Decedent both worked close to welders and grinders who produced fumes and dust. *Id.* at 75-76. He stated that the Decedent continued to work in areas where lagging was going on after 1975. *Id.* at 77. He further testified that the Decedent worked around a great deal of dust while taking paint off ships using a "needle gun." *Id.* at 79-80. On cross examination, H. G. appeared somewhat confused when he insisted that he and the Decedent continued to be exposed to asbestos dust into the 1990s, shortly before his retirement, though he could not remember the numbers of the boats that they worked on. *Id.* at 83-85. He also described the Decedent as "six, one, six two," with no facial hair which is inconsistent with the Claimant's testimony that the Decedent had a moustache while working at EB. *Id.* at 87; 30-31. H. G. testified that was represented by the Claimant's attorney on a workers' compensation claim and that he had been awarded benefits for an asbestos-related disability. *Id.* at 68, 86.

B. Medical History

The Decedent had a lengthy medical history which is summarized below. In 1986, Dr. Martin Cheniack, who was Director of the Occupational Health Clinic at the Lawrence and Memorial Hospital in New London, Connecticut, reported that the Decedent's chest x-ray showed bilateral pleural plaques which he described as a "marker of asbestos exposure, but not actually indicating decreased lung function." ALJX 15 at 4-5. In a follow-up report dated February 1, 1988, Dr. Cherniack confirmed that the Claimant had been diagnosed with "benign pleural disease secondary to exposure to asbestos." CX 11 at 1. However, by 1989, the Decedent began to show signs of reduced lung volume and a reduced ability to exhale quickly. EX 1 at 2. Other laboratory tests from this period show heightened cholesterol, sinusitis, and a normal chest x-ray. *Id.* In early 1995, a chest x-ray showed signs of early chronic obstructive pulmonary disease ("COPD"). *Id.* at 3. The Decedent also had signs of vascular disease in his peripheral arteries. *Id.* A second round of pulmonary function tests in 1996 yielded results "consistent with obstructive lung disease." *Id.* An x-ray taken on May 7, 1996 showed progression of Decedent's COPD. *Id.*

In September of 1999, the Decedent was diagnosed by his treating pulmonary specialist, Stephen L. Matarese, D.O., with a "significant respiratory impairment" and "significant emphysema and obstructive airways disease." ALJX 15 at 5-6. Dr. Matarese noted that the Decedent had a history of working closely with asbestos and exposure to dust and fumes from other trades, and he stated that the Decedent's pulmonary function studies showed obstructive physiology consistent with emphysema but also some restrictive processes suggestive of pneumoconiosis. *Id.* at 6. Dr. Matarese further noted that a CT scan showed no evidence of any pleural disease but parenchymal scarring in the lingula and also in both apices." *Id*. Dr. Matarese concluded that the Decedent had pulmonary disease aggravated by work exposures and parenchymal scarring directly related to asbestos exposure. *Id.*

In December of 1999, the Decedent was examined at EB's request by John A. Pella, M.D., another pulmonary specialist, who diagnosed "chronic obstructive pulmonary disease, predominantly pulmonary emphysema" caused primarily by cigarette smoking. ALJX 15 at 8. Dr. Pella further stead that '[i]t is probable that this condition was aggravated or caused in part by his exposures to dust, fumes and smoke exposures at Electric Boat." *Id.* At the same time, Dr. Pella wrote that he found no evidence asbestos- related occupational lung disease. *Id.*

On October 22, 2002, Dr. Matarese wrote to Joseph Dotolo, M.D. who was the decedent's primary care physician, that a chest x-ray in April showed numerous nodular densities and pleural thickening on the left as well as emphysema, and that pulmonary function studies showed moderate to severe abnormalities consistent with emphysema. CX 8 at 6. In April of 2004, Dr. Matarese reported that the Claimant's chest x-ray continued to show pleural thickening, and his assessment was (1) underlying dyspnea secondary to emphysema and COPD, (2) chronic interstitial fibrotic changes from previous occupational exposure to asbestos, and (3) peripheral vascular insufficiency. *Id.* at 3. In his final report of October 25, 2004, Dr. Matarese's assessment was (1) severe COPD, (2) asbestosis, (3) pleural thickening and (4) peripheral vascular insufficiency. *Id.* at 1.

On December 29, 2004, the Decedent was admitted to the Westerly Hospital under the care of Dr. Dotolo with complaints of abdominal and back pain after a CT scan of the abdomen revealed diffuse metastatic lesions on the liver. CX 6 at 2-3. Indeed, the liver metastasis was so advanced that consulting gastroenterology specialist Pamela Jo Connors, M.D. described the CT as showing a "liver that is almost completely replaced with low attenuation lesions." *Id.* at 5. In her initial consultation report on December 29, 2004, Dr. Connors stated that the Decedent's family history of colon cancer (*i.e.*, a brother reportedly died at age 56 from metastatic colon cancer) placed him at great risk for colon cancer warranting colonoscopy, and she stated that the list of potential sources for the Decedent's metastatic disease "would certainly include esophageal, lung and colon cancer with colon cancer being the most likely." *Id.* at 14. She then discussed these possibilities and noted that the Decedent's test results argued against colon cancer, leading her to comment that "given his history of asbestos exposure and smoking a lung malignancy with metastatic disease to the liver and possibly to bone . . . may be the most likely diagnosis." *Id.* A CT scan of the Decedent's chest was performed on December 30, 2004, and Dr. Connors reported that the scan showed a left lower lobe mass suspicious for neoplasm as well as evidence of metastatic disease involving the left hilar region, liver, spleen and celiac

region. *Id.* at 7, 19. In a post-discharge report dated January 6, 2005 in which she discussed the results of the chest CT and the negative colonoscopy (CX 6 at 7), Dr. Connors stated that it was her impression that the Claimant appeared to have metastatic lung cancer. *Id.* at 9. The Decedent was discharged on January 2, 2005 with instructions to return for a liver biopsy. *Id.* at 1.[3]

The Decedent returned to Westerly Hospital emergency room on January 6, 2005 at nine in the evening. CX-7 at 1. Nursing notes indicate that he was experiencing abdominal pain and had difficulty breathing, but he could answer questions. *Id.* at 4. However, he quickly became unresponsive and was pronounced dead at 9:29 p.m. *Id.* The emergency room physician, Christopher M. Lerach, M.D., completed the death certificate and listed "end stage adenocarcinoma – unclear primary" as the immediate cause of death. CX 2.

C. Medical Opinions

1. Dr. Matarese

In addition to his extensive treatment records, Dr. Matarese submitted a letter to the Claimant's attorney dated November 3, 2005 in which he stated that he had reviewed the Decedent's death certificate and medical records. CX 1. He stated that it appeared that the Decedent had a "primary lung carcinoma with metastasis." *Id.* He concluded, "It is of my medical opinion that it is more probable than not that his long term exposure to asbestos was a significant factor in the development of his lung carcinoma and in fact, hastening his death." *Id.*

2. Susan M. Daum, M.D.

In a report for the Claimant's attorney dated May 5, 2006, Dr. Daum, a specialist in environmental and occupational medicine, reviewed the Decedent's medical records and deposition testimony. CX 10. Dr. Daum noted that pleural thickening was first diagnosed in 1986. *Id.* at 1. She further noted that a CT scan in 1999 showed evidence of interstitial fibrotic change based on the presence of parenchymal banding and that a 2002 chest x-ray showed bilateral pleural thickening with a nodular appearance. *Id.* at 3-4. Dr. Daum also did a B-reading of an April 10, 2003 chest x-ray which she classified as positive for pleural plaques consistent with pneumoconiosis. *Id.* at 12.[4] She also commented on the presence of diffuse increased bronchovascular lung markings, flattened diaphragms, increased retrosternal space, and coarse fibrotic markings in the left mid and lower lung zones. *Id.* at 13. She stated that it is her opinion that the Decedent died of "bronchogenic (lung) cancer" which was the primary tumor and the "most likely source of his widely metastatic disease which caused his death." *Id.* at 10. She further stated that it is her opinion that the Decedent's occupational exposure to asbestos, paint fumes, solvent fumes, grinding dust, silica and other dusts, fumes and irritants at the Electric Boat Shipyard were significant contributing factors, along with his personal addiction to cigarettes [sic] smoking." *Id.* It was further Dr. Daum's opinion that the Decedent

[3] It appears that no liver biopsy or biopsy of the left lobe mass was ever performed.

[4] The April 10, 2003 chest x-ray was originally interpreted by Paul D. Cardi, M.D. who noted "[a]reas of bilateral pleural thickening . . . having a benign appearance without significant change from prior study of 5-10-02." EX 8 at 112. Dr. Cardi's impression was COPD with minor acute changes." *Id.* He mad no mention of pleural plaques.

had asbestosis of the lungs and pleura, chronic obstructive pulmonary disease and mild emphysema. *Id.* She added that based on the Decedent's severe pulmonary obstructive impairment and the rather mild emphysema described in the CT scan, it is probable that some of the Decedent's abnormal diffusing capacity were due to pulmonary asbestosis, and she commented that the diagnostic features of pulmonary asbestosis are known to be obliterated radiographically by the presence of COPD and emphysema. *Id.* Dr. Daum continued that it is well-established that all types of lung cancer are increased in asbestos-exposed populations, that the distribution of the lung cancer types in asbestos-exposed populations is that same as that which occurs in the general population, asbestos-related lung cancers do not appear clinically or pathologically different from "spontaneous" cancers arising in the general population or from those induced by other carcinogens such as cigarettes, and that there is no particular location of the cancer which rules out a contribution of asbestos-carcinogenesis. *Id.* Regarding asbestos-carcinogenesis, Dr. Daum explained,

> The fibrosis of the lung parenchyma caused by asbestos particles is mediated by a reaction of asbestos with inflammatory cells causing the expression of cellular hormones (called cytokines). Cancers are caused by asbestos fibers acting on the genetic material in the nucleus of the cell lining the bronchial tubes. Asbestos has been shown to cause changes in the genetic material (chromosome and the DNA which composes chromosome). These changes in the cell-genetic material are known to be the sort of changes associated with the initiation and promotion of cancer — the development and increasing genetic abnormality which turns normal cells into cancer cells. The process of fibrosis and cancer formation occur with different dose-response levels, a fact recognized by the most recent asbestos-exposure standard of OSHA (1984), which lowered the permissible exposure from 1 fiber/cc TWA (to prevent asbestosis) to 0.1 fiber/cc (to reduce the incidence of lung cancer) to I case/10,000 for workers exposed for 40 years.

Id. at 10-11. Dr. Daum stated that the incidence of lung cancer among smokers who are exposed to asbestos is five to seven times in excess of the incidence which occurs among smokers in the general population who are not exposed to asbestos and, further, that the risk of developing lung cancer does not decrease as rapidly after smoking cessation in asbestos-exposed individuals as it does for smokers who were not exposed to asbestos. *Id.* at 11. For these reasons, Dr. Daum concluded that "exposure to asbestos was a significant contributing factor" in the development of the Decedent's lung cancer. *Id.*

Dr. Daum testified at a deposition taken on June 2, 2006. CX 12. She graduated from the Cornell University College of Medicine and completed her residency at Mount Sinai Medical Center in New York. *Id.* at 6-7, 31. She is board-certified in internal and preventative (occupational) medicine, and she is a certified B-reader. *Id.* at 97; Deposition Exhibit 1 (*Curriculum Vitae*).[5] Dr. Daum has extensive experience in the field of occupational medicine,

[5] A certified "B-reader" is a physician who has demonstrated designated levels of proficiency in assessing and classifying x-ray evidence of pneumoconiosis by successful completion of an examination conducted by the United States Public Health Service. 42 C.F.R. §37.51; *Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 146, n. 16 (1987), *rehearing denied*, 484 U.S. 1047 (1988).

both as a physician and researcher, and she worked with Dr. Irving Selikoff at Mount Sinai from 1971 to 1979 on several studies of asbestos workers including a survey of mortality rates for workers at EB's Groton Shipyard. *Id.* at 7-9, 31-34. In 1979, she established her own preventative medicine practice which was devoted to screening workers for occupational disease, primarily on referral from unions and lawyers. *Id.* at 8, 33. Dr. Daum continues to practice preventative or occupational medicine, but she has not practiced internal medicine since 2000. *Id.* at 97-98. She is a co-author of a book entitled "Work is Dangerous to Your Health" which was published in 1971 and contains a statement in the forward that the authors "do not pretend to be unbiased." *Id.* at 101-102. She explained that the context of that statement was her advocacy for passage of the federal Toxic Substances Control Act. *Id.* at 102.

Dr. Daum testified that asbestos particles, also commonly called fibers, can be dangerous when inhaled. CX at 10. She explained that due to their small size, frequently less than ten microns, the asbestos fibers can elude the body's protective mechanisms and penetrate deep into the lung, ending up near the alveolar ducts. *Id.* at 10-11. When this happens, the body summons macrophages, white blood cells, to repair the damage. *Id.* at 11. The macrophages release chemicals, called cytokines, which initiate scaring in the form of small airways lesions that can cause obstructive airway disease. *Id.* She stated that asbestosis is known for this effect. *Id.* She further testified that the same chemicals which cause scarring in the alveolar ducts also cause inflammation and scarring in the "meat" of the lung, the interstitial space. *Id.* at 12. This interstitial scarring can cause a decrease in the number of blood vessels available for oxygen exchange, and can adversely affect the rate at which carbon dioxide is replaced by oxygen in the blood, called diffusion capacity. *Id.* Interstitial scarring also causes the lungs to become stiff, resulting in a reduction in total lung capacity which is measured as "forced vital capacity." *Id.* at 13. Additionally, Dr. Daum testified that asbestos particles can be absorbed by the pleura, a sac surrounding the lung, producing scarring known as pleural thickening. *Id.* at 13-14. She explained that pleural plaques are an asbestos-specific progression of pleural thickening that can cause restrictive lung disease. *Id.* at 15.

In addition to causing scarring, Dr. Daum explained that asbestos fibers are carcinogenic. CX-12 at 17. She said that there are three mechanisms by which asbestos can either cause cancer or aid in causing cancer: (1) trans-section of DNA which involved cutting DNA and moving it to another chromosome; (2) causing aneuploidy which is an abnormal number of chromosomes in a cell; and (3) inflammation-related mutagenesis in which strong oxidants, known as super oxide radicals, form at the site of inflammation and may be an additional contribution to lung cancer. *Id.* at 17-18. Dr. Daum further testified that cigarette smoke causes lung as well as other cancers and that asbestos exposure and tobacco smoke act in an interactive or "synergistic" manner in which the effect of one multiplies the effect of the other. *Id.* at 18-20. She asserted that the effects of both carcinogens are "interdigitated" and that they cannot, when both are present, be separated so as to allow a physician to conclude that one, but not the other, is the cause of a cancer. *Id.* at 20-21.

According to Dr. Daum, the latency period from first asbestos exposure to development of asbestosis or asbestos-related cancer ranges from ten to thirty years dependent on genetic factors of susceptibility to carcinogens that are not fully understood. CX12 at 21-23. She continued that the development of lung cancer and asbestosis are separate events and, while

correlated, neither is necessary for the other. *Id.* at 23. Dr. Daum also pointed out that interstitial fibrosis may be present pathologically but not be detected on reading of a chest x-ray or CT scan, so that absence of a finding of asbestosis in a chest x-ray or CT scan report does not exclude the presence of the condition. *Id.* at 23-24. Dr. Daum testified that the excess of lung cancer among people exposed to asbestos does not depend on the presence of asbestosis, and she added that asbestos causes cancer of a wide range of organs including the mouth, pharynx, larynx, lung, bronchogenics, alveoli, hypopharynx, esophagus, stomach, colon, rectum and, in rare cases, the pancreas, kidneys and biliary tract. *Id.* at 23-24. She said that while it takes more asbestos exposure to cause lung cancer than to cause asbestosis, the presence of pleural plaques increases the chance of developing lung cancer by three times. *Id.* at 25. She also stated that there is no "safe" type of asbestos as all categories are known to cause asbestosis and cancer. *Id.* at 28-29.

With regard to her report on the Decedent, Dr. Daum testified that she had sufficient medical records to support a differential diagnosis of asbestos-related pleural disease, chronic obstructive pulmonary disease, systolic hypertension, probable emphysema diagnosed by low diffusion capacity, possible evidence of asbestosis which she did not see, and metastatic adenocarinoma of either lung or bowel primary. *Id.* at 56-57. She stated that the presence of the lung mass made lung cancer the most likely primary site, and she stated that it was her opinion to a reasonable degree of medical certainty that the lung mass represented a lung cancer. *Id.* at 57. Dr. Daum testified that the Decedent's lung cancer resulted in his death and that the Decedent's exposures in EB's shipyard to asbestos and chromium paints were a substantial contributing factors in the development of his lung cancer and in hastening or contributing to his death. *Id.* at 61-63.[6] She further testified that the Decedent's cigarette smoking was another contributing factor and that the Claimant's smoking combined synergistically with the Claimant's exposures to asbestos, chromium and occupational dust to cause his lung cancer. *Id.* at 64. She also testify that the Decedent's underlying lung disease, which Judge Di Nardi found to be work-related, significantly affected his ability to survive. *Id.* at 65.[7] Dr. Daum stated that the Decedent did not have asbestosis, but he did have pleural plaques and pleural thickening which is "pathognomonic" for asbestos exposure and indicates that he inhaled a significant harmful dose of asbestos. *Id.* at 66.

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[6] EB's objection to the question that elicited this testimony from Dr. Daum is overruled as the question is not irrelevant, lacking appropriate foundation or impermissibly leading.

[7] EB's objection to this question is overruled as it is not materially at variance from Judge Di Nardi's findings or the medical opinions in evidence before Judge Di Nardi. Dr. Daum answered,

> I mean the underlying lung disease is going to significantly affect his ability to survive and take chemo and do well with any treatment, but his cancer was so widely metastatic, yeah, it would have affected it, because he had a lung mass removed. His lung mass is taking up room in a lung not functioning very well.

CX 12 at 65. It is noted that there is no evidence in the medical records that the Decedent's lung mass was removed. Thus, it appears that Dr. Daum misspoke when she said that the mass was removed, but she corrected this error in the next sentence when she refers to the mass as taking up room in the lung.

Regarding the Decedent's exposure to asbestos at EB, Dr. Daum testified that asbestos insulators and others breathing in the same work environment had exposures between 6 and 12 fibers per cubic centimeter which has been associated with an excess risk of lung cancer after two years of exposure, and she estimated that the Decedent's exposure places him well above the 25 fiber-year level which would double his risk and interact further with cigarette smoke. CX 12 at 67-68.[8] She further testified that it is not necessary to have actual air quality testing data in order to make a differential diagnosis based on reasonable medical probability because "what we do in occupational medicine is know that a certain particular characteristic activity or occupational history is associated with, in a particular population, an increased risk of disease X or Y." *Id.* at 73. She pointed out that this approach was endorsed in the "Helsinki Criteria"[9] and added that "there is no question, and all the judges in this place that deals with all these cases from the shipyard know that people are exposed to significant quantities of asbestos." *Id.* at 73-74. In this regard, she asserted that it is well known and discussed in research papers, including one in which she participated, that "the conditions of work [at the EB Groton shipyard] were very dusty and contained asbestos, at least until the 70s and even into the 80s." *Id.* at 75.

Dr. Daum was asked whether it is necessary to eliminate all cofounders in order to make a diagnosis (*e.g.*, eliminating asbestos as a possible cause before concluding that smoking is a cause of lung cancer), and she responded that it is not valid scientific methodology, but demagoguery, to suggest that interactive agents such as smoking and asbestos can be eliminated to a reasonable degree of medical certainty. CX 12 at 76-77. Finally, she testified that the Decedent required medical treatment for his lung cancer which was related to his asbestos exposure. *Id.* at 77.

On cross-examination by EB's attorney, Dr. Daum confirmed that there was no pathology done on the Decedent's lung mass or liver, but she stated that scientific papers show that 95 percent of patients, who have cancers of unknown primary with a lung mass, have lung cancer,

[8] In this line of questioning, the Claimant's attorney attempted to obtain an estimate from Dr. Daum as to the amount of asbestos fibers the Decedent would have been expected to inhale based on assumptions regarding fiber concentrations and lung capacity. CX 12 at 69-72. In response, Dr. Daum testified that the Decedent would have been exposed to approximately one billion fibers per hour "[i]f the calculations are right." *Id.* at 72. However, the calculations do not appear to have been right. Counsel had Dr. Daum assume a fiber density of 10 fibers per cubic centimeter, and a lung capacity of six liters or 6,000 cubic centimeters. *Id.* at 71. Based on these assumptions, counsel then miscalculated by two orders of magnitude in estimating that the number of fibers inhaled per breath would have been six million. *Id.* Ten fibers per cubic centimeter times 6,000 cubic centimeters is 60,000 fibers, not six million. Dr. Daum then estimated that the Decedent's respiratory rate while working would have been approximately 20 breaths per minute, and she concurred with counsel's suggestion that the Decedent would have inhaled "120 million fibers per second [sic]" and "close to a billion fibers an hour." *Id.* at 72. The correct estimates would be as follows: 60,000 fibers per breath times 20 breaths per minute = 1.2 million fibers per minute; 1.2 million fibers per minute times 60 minutes per hour = 72 million fibers per hour; 72 million fibers per hour times 8 hours per day = 556 million fibers per eight-hour day. Thus, Dr. Daum's estimate of close to one billion fibers per hour is more than ten times the correct estimate.

[9] The "Helsinki Criteria" or "consensus report" is appended to the transcript of Dr. Daum's deposition as Claimant's Exhibit A, and was published at 23 Scand. J. Work Environ. Health 311 (1997). The Helsinki researchers concluded that "[c]umulative exposure, on a probability basis, should thus be considered the main criterion for the attribution of a substantial contribution by asbestos to lung cancer risk." *Id.* at 314. The report further states that "relative risk is roughly doubled for cohorts exposed to asbestos fibers at a cumulative exposure of 25 fiber-years or with an equivalent occupational history, at which level asbestosis may or may not be present or detectable." *Id.*

especially when the cancer is widely metastatic. *Id.* at 79. She agreed that without an autopsy, one could not determine with "absolute certainty" that the lung mass was the primary site of the Decedent's cancer, but she stated that it is her opinion on the basis of reasonable medical causation that the Decedent's pulmonary mass was causing the widely metastatic cancer. *Id.* at 80. Dr. Daum testified that other candidates for the primary site would be gastrointestinal, such as esophageal, colon or pancreas, of the head and neck. *Id.* at 80-81. She confirmed that she did not read the Decedent's chest x-ray as showing parenchymal asbestosis or interstitial disease, but she did find that he had pleural thickening and pleural plaques which she referred to as "pleural asbestosis" in her report and which are caused by asbestos exposure through the same pathological process that creates asbestosis. *Id.* at 82-83. She also testified that x-rays and CT scans to a lesser extent underestimate the presence of asbestosis, but that she could not be certain that the Decedent had asbestosis without pathology. *Id.* at 84. Dr. Daum rejected a suggestion that Decedent's abdominal obesity or "beer belly" would adversely affect the results of is pulmonary function testing, at least, not to the degree seen. *Id.* at 87. She acknowledged that the Helsinki dealt primarily with shipyards outside of the United States, and she agreed that the Decedent had a significant smoking history which was a significant contributing factor, along with asbestos and chromium, in his development of lung cancer. *Id.* at 88. She also acknowledged that she had not personally visited the EB shipyard in Groton and that her knowledge of working conditions there was, therefore, second-hand. *Id.* at 93-94. However, she stated that loose asbestos is known to be a potent cause of lung cancer and that the Decedent had described exposure to loose asbestos insulation products, so she did not need actual air samples for the Decedent since the general level of exposure at EB was "well-characterized." *Id.* at 95.

3. Milo Pulde, M.D.

Dr. Pulde provided two reports to EB, EX 1 and 9, and he testified at a deposition taken on June 28, 2006. EX 11. Dr. Pulde is board-certified in internal medicine and is a full-time practicing internist and attending physician at the Brigham and Women's Hospital in Boston. EX-11 at 4-5; EX-2 at 29. He is also an Assistant Professor at Harvard Medical School. *Id.*

In his initial report which is dated March 19, 2006, Dr. Pulde reviewed the Decedent's medical records and his deposition testimony. EX 1 at 1. He concluded that the Decedent had "[p]resumptive metastatic tobacco related nonsmall cell lung cancer with left lower lobe mass by chest CT 12/30/04 and metastases to the liver, spleen and celiac lymph nodes by abdominal CTs 12/27/04 and 12/30/04." *Id.* at 7. He found no evidence in the Decedent's medical records for asbestos-related pleural plaques, parenchymal asbestosis or any asbestos-related malignancy. *Id.* He stated that "[t]here is clinical and objective evidence that supports a diagnosis of substantial and long duration tobacco abuse . . . that resulted in both tobacco related chronic obstructive pulmonary disease . . . and presumptive metastatic stage IV tobacco related nonsmall cell lung cancer that presented as a left lower lobe mass . . . and diffuse metastases to the liver, spleen, and celiac lymph nodes . . . and, most likely culminating, in death at an indeterminate time." *Id.* at 8.[10] Dr. Pulde further stated that the evidence "supports a diagnosis of occupational exposure to dust, fumes, vapors, and gasses 01/24/63 to 12/23/98" but not any occupational disease. *Id.* He

[10] Although Dr. Pulde did not review the death certificate at the time of his initial report, it is unclear why he referred to "death at an indeterminate time" because he did review the Westerly Hospital records which clearly state that the Decedent was pronounced dead at 2129 (9:29 p.m.) on January 6, 2005. CX 7 at 4.

thus concluded that to a reasonable degree of medical certainty, the Decedent's "pulmonary diagnosis is consistent with tobacco related chronic emphysema and presumptive tobacco related non-small cell lung cancer." *Id.* at 9. He also concluded to a reasonable degree of medical certainty that the Decedent "did not fulfill the criteria for a clinical diagnosis of asbestos related pleural plaques, parenchymal asbestosis, or an asbestos related lung cancer." *Id.* Dr. Pulde conducted a comprehensive review of the scientific literature on asbsestos exposure and development of asbestos-related disease. *Id.* at 15-23. He concluded that the scientific literature establishes: (1) that lung fibrosis of many causes in addition to asbestosis is associated with an increased risk of lung cancer; (2) the synergism between asbestos and cigarette smoke is a synergism between cigarette smoking and asbestosis and not just asbestos exposure alone; (3) the site of origin and cell type of lung cancer is not a reliable indicator of asbestos causation; (4) in asbestos inhalation experiments, animals only develop excess lung tumors when fibrosis is also produced; (5) pleural plaques have not proven to be reliable markers of increased risk of lung cancer. *Id.* at 23. Because he found that the Decedent did not fulfill the criteria for a diagnosis of parenchymal asbestosis (*i.e.*, fibrosis), Dr. Pulde concluded that he "lacked the prerequisite criteria for attribution, in part, of his tobacco related lung cancer to asbestos exposure." *Id.* at 26. He added that the Decedent's lung cancer could be fully explained on the basis of his lengthy smoking history and documented COPD, and he said that it is not necessary to additionally implicate asbestos exposure as a factor. *Id.* Thus, he stated that there is no evidence that the Decedent's occupational exposures at EB "influenced the natural history or affected the outcome of his tobacco related lung cancer or accelerated his probable ultimate death from progressive metastatic lung cancer." *Id.*

In his second report, Dr. Pulde reviewed additional records including the death certificate, office notes of Dr. Matarese from 1999 through November 3, 2005,[11] Dr. Dotolo's admission note of December 29, 2004 and Judge Di Nardi's decision. CX 9 at 2. On the basis of this additional information, Dr. Pulde altered his diagnosis to (1) progressive adenocarcinoma of unknown primary ("AUP") with left lower lobe mass and metastases to the liver, spleen, and celiac lymph nodes, (2) tobacco abuse with COPD and emphysema, and (3) no evidence of asbestos-related pleural plaques, parenchymal asbestosis or asbestos-related malignancy. *Id.* at 7. Dr. Pulde notes that cancer of an unknown primary site ("CUP") represents three to five percent of all malignancies and that a "diagnosis of CUP can only be made if the histology of the tumor is not consistent with the known tumors of the organ from which the biopsy was taken." *Id.*[12] Dr. Pulde stated that "a definitive determination of the primary site of [the Decedent's] metastatic AUP cannot be made with any certainty" without an autopsy even though he acknowledged that the Decedent's smoking history placed him at increased risk for lung cancer and that there was "no definitive evidence" of a primary cancer at any other potential site. *Id.* at 11-12. Moreover, even if he were to accept his former diagnosis of presumptive metastatic adenocarcinoma of the lung, Dr. Pulde concluded that 'there is no relationship between [the Decedent's] lung cancer and his occupational exposures based on his exposure history and

[11] Dr. Matarese's office notes end in October of 2004. CX 8. His letter to the Claimant's attorney addressing the cause of the Decedent's death is dated November 3, 2005. CX 1.

[12] As discussed above, no biopsy was ever taken from the Decedent's lung mass or his liver.

failure to demonstrate physiological, radiographic, or pathological evidence of parenchymal asbestosis or other types of occupationally induced fibrotic lung disease." *Id.* at 12.

Dr. Pulde testified at his deposition that the Decedent abused tobacco, smoking at least a pack per day for forty-five years and that his occupational exposure to asbestos was "infrequent and indirect." EX 11 at 7-8. He noted that pulmonary function tests demonstrated chronic obstructive pulmonary disease but lacked evidence of "restrictive lung disease consistent with . . . parenchymal asbestosis." *Id.* at 8.[13] He contradicted Dr. Matarese's diagnosis of asbestosis, stating that there is no radiological evidence of asbestosis, in conjunction with the pulmonary function tests showing no signs of restrictive lung disease. *Id.* at 9. Without a diagnosis of parenchymal asbestosis, Dr. Pulde testified, it is difficult to point to work-related asbestos exposure as a cause of Decedent's adenocarcinoma of unknown primary origin ("AUP") since the presence of asbestosis is a prerequisite to the diagnosis of an asbestos-related malignancy. *Id.* at 10-11. Dr. Pulde indicated that he had reviewed the Decedent's x-ray and CT scan films in a May 5, 2006 report which is appended to the deposition transcript as Exhibit 1. *Id.* at 11. He testified that his review showed pleural thickening but no evidence of pleural plaques which appear different radiographically and are not necessarily specific for asbestos exposure but rather any past inflammatory condition of the lung such as pneumonia, pleurisy and rib fracture. *Id.* at 12-13, 27-28.[14] Dr. Pulde reiterated his revised opinion that the Decedent's cause of death was the "consequence of progressive . . . adenocarcinoma of an unknown primary." *Id.* at 13-14. He testified that the origin of the cancer was unknown because neither a biopsy nor an autopsy was performed. EX-11, 14. He further explained that it is difficult to make a determination as to the primary cause of the cancer without tissue studies because non-pathology evidence such as medical history or x-rays has been shown to be inaccurate between 30 and 44 percent of the time. *Id.* at 15-16. Thus, he said that "given [the Decedent's] tobacco consumption and x-ray . . . a primary lung cancer is a good probability, but I can't say that to a reasonable degree of medical certainty." *Id.* at 16. He explained that he changed his diagnostic opinion from presumptive metastatic lung cancer to adenocarcinoma of unknown primary because he had mistakenly assumed that a biopsy had confirmed the type of tumor and, more importantly, he had concluded that the Decedent's colonoscopy was not "negative" for colon cancer as reported by Dr. Connors but "non-diagnostic" due to inadequate preparation and the presence of stool which would have obscured the primary colon cancer. *Id.* at 17-18.[15] For these reasons, and based on his review of the medical records and literature, he concluded that a "definitive determination as to the primary cancer responsible for [the Decedent's] death cannot be made with any medical certainty in the absence of a tissue diagnosis either by biopsy or autopsy" and that there is no evidence that the Decedent's occupational exposures at EB caused or contributed to his cancer or accelerated his death. *Id.* at 19-20. Dr. Pulde testified that even if the court were to accept Dr. Daum's opinion that Decedent had a primary lung cancer, his opinion on death causation would not change

[13] Dr. Matarese stated in the 1999 report quoted by Judge Di Nardi that the Decedent's pulmonary function studies showed some "restrictive process suggestive of pneumoconiosis" in addition to COPD consistent with emphysema. ALJ Di Nardi Decision and Order at 6.

[14] There is no evidence that the Decedent was ever diagnosed with any of these other conditions associated with pleural plaques.

[15] Dr. Connors' colonoscopy report, which is not in evidence, was not one of the additional medical records that Dr. Pulde reviewed in his May 1, 2005 report.

because the Decedent "did not fulfill the criteria for a clinical diagnosis of parenchymal asbestosis or asbestos-related pleural plaques, and, therefore, lacked the prerequisite for the attribution of his tobacco-laden lung cancer . . . at least in part to asbestos exposure." *Id.* at 20-21. He further testified that even if the Decedent did have pleural plaques, that alone would not be enough to relate lung cancer to exposures at EB. *Id.* at 23. In this regard, he explained that while he respects Dr. Daum, her opinion that there is an increased risk for lung cancer with pleural plaques alone is contradicted by the "aggregate" of medical literature, and he cited several studies between 1979 and 1994 in support of his opinion. *Id.* at 24-25.

Dr. Pulde also disagreed with Dr. Daum's opinion that the Decedent's occupational exposures to solvents and paints contributed to his lung disease, lung cancer and death, noting that the medical literature does not consider any of these substances to be lung or pulmonary carcinogens. EX 11 at 21-22. On this question, he concluded, that "based on the failure to demonstrate either physiologic [or] radiographic evidence of asbestosis based on the type of exposures enumerated by Dr. Daum, there is no evidence that [the Decedent's] litany of occupational exposures . . . either caused a presumed lung cancer or accelerated the presentation of that lung cancer and influenced the outcome of that lung cancer on [the Decedent's] death." *Id.* at 22. He also stated that "there are no physiologic, radiographic, or even pathologic studies which can be used to determine the frequency or intensity of exposure or cumulative exposure" of any of the non-asbestos substances that the Decedent was exposed to while employed at EB. *Id.* at 23.

Additional areas of disagreement between Drs. Daum and Pulde involve the questions of whether there is a minimum threshold for asbestos exposure before it can be causally linked to lung cancer and whether the Decedent's occupational lung disease, for which he was awarded compensation by Judge Di Nardi, contributed to his death. Dr. Pulde testified that the asbestos exposure threshold issue is "somewhat controversial . . . but nevertheless at exposures less than 25 fibers per cc per year . . . there is no increased risk of lung cancer with asbestos exposure." *Id.* at 26. He further testified that the Decedent did not die of pulmonary disease and that pulmonary disease did not contribute to his death. *Id.* at 31. Rather, he said that the Decedent's death was due to "a rapidly progressive malignancy that . . . replaced his entire liver." *Id.* Finally, Dr. Pulde disagreed in part with Dr. Daum on the synergistic relationship between asbestos and cigarette smoke, asserting that the weight of medical literature establishes that the synergism comes into play only in the presence of asbestosis or asbestos-related fibrosis, not asbestos exposure alone: "[o]ne has to prove the presence of not only asbestos exposure but fibrosis secondary to that asbestos exposure before we can indicate that asbestos exposure in the development of a tobacco-related lung cancer." *Id.* at 32-33.

On cross examination, Dr. Pulde testified that lung cancer is the most common type of internal cancer, and he stated that he agreed with Dr. Connors' initial assessment that Decedent likely had either colon or lung cancer as his primary cancer. EX-11 at 35-37.[16] However, he disagreed with Dr. Connor's statement that the Decedent's colonoscopy was negative, and he instead asserted that the colonoscopy was "non-diagnostic and didn't completely exclude a colon cancer since even with adequate prep, one can miss a colon cancer." *Id.* at 38. In support of this view, he noted that Dr. Connor's colonoscopy report which is not in evidence indicated

[16] Dr. Pulde indicated that skin cancer is the most common malignancy found in humans. EX 11 at 35.

"extremely poor" preparation with fecal matter scattered throughout the colon, that the Decedent's "CA" was elevated which is associated with colon cancer, that the Decedent was at familial risk for colon cancer, and that he himself has had several patients with metastatic colon cancer despite negative colonoscopies. *Id.* at 38-39. The question of the primary source aside, Dr. Pulde agreed that cancer played a significant role in the Decedent's death. *Id.* at 39-40.

Dr. Pulde further testified that there is a correlation between exposure and lung burden of asbestos and that the risk of asbestos-related disease and cancer "correlates, indirectly at least, with the asbestos exposure." EX 11 at 41. He agreed that a person's risk of developing asbestos-related disease increases with exposure, though he said that it is not "inevitable" that they will develop disease. *Id.* at 42-43. He also agreed, albeit grudgingly and only after forcing counsel to repeat his question multiple times, that if the Decedent had pleural plaques, the plaques would indicate that asbestos had indeed migrated through his lungs and into the pleura and, therefore, that he had sustained an internal dose of asbestos. *Id.* at 43-44. Dr. Pulde was then questioned about his reference to "25 fibers per cc per year" and said that the Decedent's "exposure in fiber years would have to exceed 25 for him to have the potential of an asbestos-related pulmonary condition" and that the 25 fiber year threshold is a "sort of minimal burden . . . below which an asbestos-related condition is unlikely." *Id.* at 45.

Dr. Pulde acknowledged that if someone consumed a large amount of tobacco over their lifetime and developed lung cancer without COPD, emphysema or any other tobacco-related disease, he would still presume that tobacco was a cause of the cancer. EX 11 at 46-49. He next agreed that low oxygen saturation in the blood ("SaO_2") would put someone at greater risk of a lethal cardiac event and that gasping for breath and need of supplemental oxygen is indicative of a significant reduction of blood oxygen which would place the person at greater risk of imminent death. *Id.* at 50-52.[17] Although he insisted that there is no synergy between asbestos exposure and tobacco exposure in the absence of asbestosis or "diffuse" fibrotic lung disease, he described the carginogenic effect of asbestos fibers in much the same way as Dr. Daum. *Id.* at 52-56. He stated that it is "possible, not probable" that the Decedent could have had asbestosis, as indicated by decreased diffusion capacity, that was not detectable by x-ray, but he added that he would expect to see interstitial changes by chest CT if diffusion capacity reductions of the magnitude experienced by the Decedent had been caused by asbestosis. *Id.* at 59. He further explained that if reduced diffusion capacity were asbestos-related, he would expect to see other physiologic measures of an asbestos-related condition such as total lung capacity and chest CT. *Id.* at 60. Lastly, he agreed that a study comparing x-ray with pathology results showed that a chest x-ray missed asbestos–related fibrosis in 17 percent of the studied individuals, and he commented that this is why a B-reader will not make a diagnosis of asbestos-related pulmonary disease based on chest x-rays alone because they are not as sensitive as chest CDs. *Id.* at 61.

[17] The emergency room records from the Decedent's admission to the Westerly Hospital shortly before his death on January 6, 2005 reflect that his SaO_2 was 70 percent, describe his breathing as "gasping" and indicate that he was placed on oxygen. CX 7 at 3-4. Dr. Pulde testified that a SaO_2 below 95 percent is indicative of some impairment. EX 11 at 51.

4. Raymond D. Harbison, Ph.D.

Dr. Harbison authored a report for EB dated February 9, 2006, and a redacted version of that report was admitted into evidence as EX 3. Dr. Harbison is a board-certified toxicologist who currently holds several teaching and administrative positions at universities across the country. *Id.* at 1. Dr. Harbison has published over 150 scientific articles and authored several textbook chapters on the topic of occupational toxicology. *Id.* He stated that he reviewed the Decedent's medical records and that "[s]cientific literature does not support a claim that any asbestos exposure can cause cancer." *Id.* at 3 (citing a 2004 paper by Lee *et al*., which is not in the record, as concluding that the "relationship between asbestos exposure and lung cancer is controversial."). He noted that Decedent did not have a diagnosis of asbestosis and that researchers in 1999 and 2005 concluded that asbestosis is a much better predictor for excess lung cancer risk than measures of exposure and that the question of whether lung cancer can be attributed to asbestos exposure in the absence of asbestosis is controversial. *Id.* at 4, 11. According to Dr. Harbison, the Decedent's "[a]ge, body weight, physical activity, and diet" were additional risk factors for developing lung cancer. *Id.* at 6. He stated that these other risk factors, such as dietary fat, had not been ruled out as possible causes of the Decedent's lung cancer and "[a]dditionally, specific asbestos-induced changes at levels expected to be found in the workplace of [the Decedent] at Electric Boat have not been identified . . .[n]o specific asbestos-induced injury has been recorded . . . [a]sebestos has not been properly ruled in as a cause of [the Decedent's] lung cancer. . . [and n]one of the clinical laboratory test results establish that [the Decedent] received a harmful dose of asbestos in his workplace." *Id.* at 12. Dr. Harbison thus concluded that there is no objective evidence to rule in asbestos as the cause of the Decedent's lung cancer and that "[e]ven under the minimal standards described herein, there is no scientific basis for concluding that asbestos exposure or any other exposure in the workplace . . . caused his lung cancer or any of his other cancers." *Id.*[18]

IV. Findings of Fact and Conclusions of Law

Section 9 of the Act provides for payment of compensation and benefits to survivors of an injured worker in cases where a work-related injury "causes death." 33 U.S.C. § 909. The work-related injury or disease does not have to be the sole, primary or proximate cause of death under the Act's aggravation doctrine which holds that if an injury "aggravates, exacerbates, accelerates, contributes to, or combines with a previous infirmity, disease, or underlying condition, the resultant condition is compensable . . . consistent with the maxim that 'to hasten death is to cause it.'" *Woodside v. Bethlehem Steel Corp.*, 14 BRBS 601, 603 (1982) (quoting *Avignon Freres v. Cardillo*, 117 F.2d 385, 386 (D.C. Cir. 1940)). The Claimant states that Judge Di Nardi found that the Decedent suffered work-related lung injuries consisting of chronic obstructive pulmonary disease, emphysema and asbestosis, and it asserts that EB is barred by the doctrine of collateral estoppel from contesting these issues. Claimant Br. at 11. The Claimant further contends that even if these issues had not been decided in the prior proceeding, the evidence is overwhelming that the Decedent suffered from "asbestosis and chromium lung

[18] "Minimal standards" is an apparent reference to the "preponderance of the evidence" burden placed on claimants seeking benefits under the Act.

disease which contributed to the development of his lung cancer and resulting death, and that the lung condition alone hastened death." *Id.*

A. Applicability of Collateral Estoppel

Collateral estoppel, or issue preclusion, applies where: (1) the identical issue was raised in a previous proceeding; (2) the issue was actually litigated and decided in the previous proceeding; (3) the party had a full and fair opportunity to litigate the issue; and (4) the resolution of the issue was necessary to support a valid and final judgment on the merits. *Uzdavines v. Weeks Marine, Inc.*, 418 F.3d 138, 146 (2d Cir. 2005); *Holmes v. Shell Offshore, Inc.*, 37 BRBS 27, 29 (2003). Where these elements are present, issues decided in a disability claim brought under the Act are binding on the parties in a subsequent death claim. *Taylor v. Plant Shipyards Corp.*, 30 BRBS 90, 96-97 (1996).

In the prior proceeding, the Decedent alleged that his chronic obstructive pulmonary disease resulted from his "working conditions or resulted from his exposure to and inhalation of asbestos and other injurious pulmonary stimuli in the Employer's shipyard." ALJ Di Nardi Decision and Order at 10. Therefore, the issues before Judge Di Nardi were (1) whether the Decedent's pulmonary injury was causally related to his maritime employment and (2) if so, the nature and extent of any disability. *Id.* at 3. On the first issue, Judge Di Nardi concluded that the Decedent's "daily exposure to and inhalation of asbestos dust and fibers and other injurious pulmonary stimuli, has resulted in pulmonary problems diagnosed as asbestos [sic] and chronic obstructive pulmonary disease . . . ." *Id.* at 11. In making this finding, Judge Di Nardi relied on the medical reports from Drs. Cherniack, Matarese and Pella, none of whom made a specific diagnosis of "asbestosis." Indeed, Dr. Cherniack diagnosed the Claimant with pleural plaques but no asbestos-related decrease in lung function, and Dr. Pella diagnosed COPD, predominantly pulmonary emphysema that had been aggravated or caused in part by exposures at EB, but no asbestos-related occupational lung disease. However, Dr. Matarese diagnosed both pulmonary disease aggravated by work exposures and "parenchymal scarring [sic] that is directed related to his asbestos exposure." CX 8 at 13. It thus appears that Judge Di Nardi construed the reference to asbestos-related parenchymal scarring as constituting as a diagnosis of asbestosis.

Based on the foregoing, I find that the issue raised and actually litigated and decided in the prior proceeding was whether the Decedent's COPD was related to his employment at EB. Judge Di Nardi also apparently decided that the Decedent had been diagnosed with asbestosis, but I conclude that EB did not have a full and fair opportunity to litigate this issue because the record does not show that the Decedent ever alleged that he suffered from asbestosis, and because none of the physicians made a specific diagnosis of asbestosis. A party not made aware of an issue can hardly be found to have been provided with a full and fair opportunity to litigate that issue. Moreover, Judge Di Nardi's finding that the Claimant had been diagnosed with asbestosis was not necessary to support his award of compensation which was independently supported by his finding that the Decedent's COPD and related pulmonary impairment were caused or aggravated by his EB employment. Since the final two criteria for application of collateral estoppel are not present, I find that EB is not precluded from disputing herein whether the Decedent had asbestosis. On the other hand, I find that all four criteria are met with respect

to the prior finding that the Decedent's COPD was work-related, and I therefore conclude that EB is barred from relitigating that issue in this proceeding.[19]

B. Did the Decedent's work-related COPD hasten his death?

Dr. Daum testified that the Claimant's underlying lung disease hastened his death because it significantly affected his ability to survive and take chemotherapy and any other treatment. Dr. Pulde disagreed to some extent, asserting that the Decedent did not die of pulmonary disease and that pulmonary disease did not contribute to his death. However, he agreed on cross-examination that low SaO_2 would put someone at greater risk of a lethal cardiac event and that gasping for breath and need of supplemental oxygen is indicative of a significant reduction of blood oxygen which would place the person at greater risk of imminent death. As noted above, the Decedent's medical records show that shortly before his death, his SaO_2 was measured at 70 percent, his breathing was described as gasping for air, and he was placed on supplemental oxygen. No physician has questioned Dr. Matarese's assessment that the Decedent's COPD was severe or that his shortness of breath was related to his COPD. Given the severity of the Decedent's COPD and the fact that his medical records confirm the presence of pulmonary signs and symptoms that, according to Dr. Pulde, placed him at great risk of imminent death, I find that the evidence establishes that the Decedent's underlying COPD was contributory to the extent that it acted to hasten his death on January 6, 2005. While it is clear that the Decedent's predominant medical condition was his metastatic cancer, the contributory role played by his COPD and its impairment of his pulmonary function, albeit minor in relation to the cancer, is sufficient to support an award of death benefits under the Act. *See Fineman v. Newport News Shipbuilding & Dry Dock Co*., 27 BRBS 104, 108-109 (1993) (affirming the "hastening death" standard and rejecting argument that a work-related condition must bear a "direct relationship" to death before benefits can be awarded).

C. Was the Decedent's cancer related to his employment at EB?

Though it is not necessary to reach this issue in light of my determination that the Decedent's work-related COPD hastened his death, it is the primary issue litigated by the parties and will, therefore, be addressed in the interest of completeness. Section 20(a) of the Act presumes, in the absence of substantial evidence to the contrary, that a claim for death benefits comes within the provisions of the LHWCA, *i.e.*, that the death was work-related. *Sprague v. Director, OWCP*, 688 F.2d 862, 865 (1st Cir. 1982); *Fineman v. Newport News Shipbuilding and Dry Dock Co*., 27 BRBS 104, 107 (1993). This presumption is invoked by a showing that the worker "suffered harm, and that workplace conditions or a workplace accident could have caused, aggravated, or accelerated the harm." *American Stevedoring Ltd. v. Marinelli*, 248 F.3d 54, 64-65 (2d Cir. 2001) (*Marinelli*). "If the claimant thus qualifies for the presumption, the burden shifts to the employer to rebut the presumption with substantial evidence that the alleged harmful workplace condition did not cause, contribute to or aggravate the claimant's condition." *Id.* at 65. "Finally, if the employer offers evidence sufficient to rebut the presumption, then all relevant evidence must be weighed to determine if a causal relationship has been established, with claimant bearing the ultimate burden of persuasion." *Id.*

[19] EB has not disputed Judge Di Nardi's finding that the Decedent's COPD is work-related.

1. The Claimant's *Prima Facie* Case and Invocation of the Presumption

The medical records show that Decedent had a lung mass and that cancer had metastasized to his liver and other organs. His deposition testimony, which was corroborated by the testimony of the two former co-workers that the Claimant called at the hearing, establishes that he was exposed to airborne asbestos insulation dust, chromate and other paint fumes, and welding fumes in the course of his employment at EB. While I agree with that the reliability of H.G.'s memory is suspect, especially with regard to his testimony that he and the Claimant were exposed to asbestos into the 1990s, I found E.T. to be a reliable and credible witness and find that his testimony, in conjunction with the testimony of the Decedent and Dr. Daum and the report from Dr. Cherniack, establishes that the Decedent had regular exposure to asbestos dust, much of it without effective breathing protection from 1963 until the mid-1970s.[20] Based on this evidence and Dr. Daum's testimony that asbestos exposure alone, and more so in combination with cigarette smoke, significantly increases the risk of lung cancer, I find that the Claimant has made out a prima facie case that the Decedent's cancer was related to his employment at EB. Dr. Daum also testified that the Decedent's exposure to chromium paints elevated his cancer risk.

2. EB's Rebuttal

Since the Claimant has successfully invoked the presumption that the Decedent's cancer was causally related to his occupational exposure to asbestos and/or other carcinogenic substances at EB, the burden shifts to EB to produce substantial evidence that these exposures did not cause, contribute to, or aggravate the Decedent's condition. *Marinelli*, 248 F.3d at 65. Evidence is "substantial" if it is the kind that a reasonable mind might accept as adequate to support a conclusion. *Richardson v. Perales*, 402 U.S. 389, 401 (1971); *Sprague v. Director, OWCP*, 688 F.2d 862, 865 (1st Cir. 1982). Under the substantial evidence standard, an employer does not have to exclude any possibility of a causal connection to employment, for this would be an impossible burden; it is enough that it produce medical evidence of "reasonable probabilities" of non-causation. *Bath Iron Works Corp. v. Director, OWCP*, 137 F.3d 673, 675 (1st Cir. 1998). *See also Ortco Contractors, Inc. v. Charpentier*, 332 F.3d 283, 289 (5th Cir. 2003) (rejecting requirement that an employer "rule out" causation or submit "unequivocal" or "specific and comprehensive" evidence to rebut the presumption and reaffirming that "the evidentiary standard for rebutting the § 20(a) presumption is the minimal requirement that an employer submit only substantial evidence to contrary."), *cert. denied*, 540 U.S. 1056 (2003).

EB introduced Dr. Pulde's opinion that there is no evidence that the Decedent's employment caused or contributed to his cancer or accelerated his death. Additionally, Dr. Harbison concluded that there is no scientific basis for concluding that asbestos exposure, or any other exposure in the workplace caused the Decedent's lung cancer or any of his other cancers." I find that a reasonable mind could accept these opinions from qualified experts as sufficient to support a conclusion of non-causation with respect to the Decedent's cancer. As outlined above,

[20] EB argues that E.T.'s knowledge of the Decedent and his exposures at EB are suspect because of discrepancies between his testimony and that of the Claimant on such points as whether the Decedent had a moustache, his height and hobbies. EB Br. at 15-17. In my view, these discrepancies are minor and do not materially detract from the reliability of E.T.'s testimony relating to the working conditions that he and the Decedent encountered while working together at EB between 1963 and the mid-1970s.

evidence of this caliber is substantial and sufficient to rebut the section 20(a) presumption of causation.

3. Has the Claimant proved causation by a preponderance of the evidence?

Because the presumption has been rebutted, it "falls out" of the case, and the administrative law judge must weigh all the evidence and render a decision supported by substantial evidence. *Del Vecchio v. Bowers*, 296 U.S. 280, 286-287 (1935). As the proponent of an award of benefits under the Act, the Claimant bears the ultimate burden of persuasion. *Marinelli*, 248 F.3d at 65; *Director, OWCP v. Greenwich Collieries*, 512 U.S. 267, 277-280 (1994); *John W. McGrath Corp. v. Hughes*, 264 F.2d 314, 317 (2d Cir. 1959), *cert. denied*, 360 U.S. 931 (1959).

In staking out their positions on the question as to whether the Decedent's cancer can be linked to his employment at EB, the medical experts differed on whether his diagnosis included asbestosis, pleural plaques and lung cancer. As an initial matter, I will assess whether the presence of any of these conditions is established by a preponderance of the evidence.

Asbestosis

Dr. Matarese is the only physician to diagnose asbestosis, and his brief opinion letter provides no explanation of the basis for his diagnosis. Dr. Matarese is a pulmonary specialist, but the record does not show that he has any specialized qualification in interpreting imaging studies, such as B-reader certification or board-certification in radiology. Moreover, there is no mention of asbestosis in any of the reports of chest x-rays and CT scans, and Dr. Daum, a certified B-reader who was retained by the Claimant, testified that she did not find sufficient evidence to support a finding of asbestosis. Accordingly, I find that a preponderance of the evidence does not establish that the Decedent had asbestosis.

Pleural Plaques

Dr. Cherniack reported in 1986 that the Decedent's chest x-ray showed bilateral pleural plaques. The 1986 x-ray report is not in evidence, and subsequent chest x-ray and CT scan reports in the record at CX 6, CX 8 and EX 8 do not mention pleural plaques. Dr. Pulde reviewed several x-rays and CT scans and saw no evidence of pleural plaques which he distinguished from pleural thickening. EX 11 at 11-13, 27-28. However, Dr. Daum re-read the Decedent's April 10, 2003 chest x-ray, which had been initially interpreted by a radiologist as showing bilateral pleural thickening (EX 8 at 112), as positive for pleural plaques consistent with pneumoconiosis. CX 10 at 12. In view of the fact that EB has not demonstrated that Dr. Pulde has any particular expertise in interpreting radiological studies, and in the absence of any evidence establishing the qualifications of the radiologists who interpreted the Decedent's chest x-rays and CT scans, I find it reasonable to give greater weight to the interpretation by Dr. Daum based on her B-reader certification. *See Woodward v. Director, OWCP*, 991 F.2d 314, 316 n.4 (6th Cir. 1993) (Black Lung Benefits Act case noting that it is appropriate to give more weight to the interpretation of a B-reader because of their expertise). Therefore, I find that the weight of evidence establishes that the Decedent had pleural plaques.

Lung Cancer

There is a sharp conflict in the medical opinions on the question of whether the primary source of the Decedent's fatal metastasizing cancer was the mass seen in his lung. Dr. Matarese stated that it was his opinion from reviewing the medical records and death certificate, on which Dr. Lehrach listed "end stage adenocarcinoma of unclear primary" as the cause of death (CX 2), that the Decedent had primary lung carcinoma with metastasis. CX 1. Dr. Connors, the gastroenterologist who treated the Claimant during his admission to the Westerly Hospital in the days before his death, initially felt that the primary cancer source was either the lung or colon, but her final impression was apparent metastatic lung cancer after she reported that her colonoscopy was negative. CX 6 at 9. Dr. Daum concurred with this assessment, testifying that although one could not be absolutely certain since pathology studies were not conducted, it was her opinion to a reasonable degree of medical certainty that the Decedent's primary cancer source was the mass in his lung given the fact that studies have shown that 95 percent of patients, who have cancers of unknown primary with a lung mass, have lung cancer, especially when the cancer is widely metastatic. CX 12 at 79-80. Dr. Pulde at first joined the medical consensus for a primary lung cancer when he diagnosed the Decedent with "[p]resumptive metastatic tobacco related nonsmall cell lung cancer with left lower lobe mass by chest CT 12/30/04 and metastases to the liver, spleen and celiac lymph nodes by abdominal CTs 12/27/04 and 12/30/04." EX 1 at 7. However, he jumped ship in his supplemental report wherein he stated that after reviewing additional records (*i.e.*, the death certificate and Dr. Matarese's office notes), he changed his cancer diagnosis to "progressive adenocarcinoma of unknown primary (AUP)." CX 9 at 7.[21] For the reasons discussed below, I am not persuaded that Dr. Pulde's doubts require a finding that the evidence falls short of establishing the presence of a primary lung cancer.

First, Dr. Pulde relied heavily on the cause of death listed in the death certificate, but Lehrach did not diagnose an adenocarcinoma of "unknown primary." Rather, he listed adenocarcinoma of unclear primary as the cause of death. What Dr. Lehrach meant by his use of the term "unclear" is a matter of speculation because he was never deposed or even asked for an opinion letter, but I find it reasonable in the context of the medical records to interpret his choice of the term "unclear" as most likely reflecting his recognition that no biopsy or autopsy had been performed to conclusively identify the primary cancer site, not a diagnosis of an adenocarcinoma of unknown primary. It also strikes me as medically unsound to credit a few words on a death certificate from and emergency room physician who appears to have treated the Decedent for no more than a few minutes over the extensive reports from the treating gastroenterologist.[22] Second, Dr. Pulde relied on his own interpretation of Dr. Connors' colonoscopy report to override Dr. Connors' negative finding and conclude that the study was "non-diagnostic" and insufficient to exclude a primary colon cancer. Dr. Pulde is board-certified in internal medicine, but there is nothing in the record to show that he has any expertise in interpreting colonoscopy

[21] In his supplemental report, Dr. Pulde also reviewed Judge Di Nardi's decision which found the Decedent's COPD to be related to asbestos and other exposures at EB. He has not indicated that Judge Di Nardi's decision played any role in the transformation of his diagnosis away from lung cancer.

[22] The ER records indicate that the Decedent arrived by ambulance at 2100 (9:00 p.m.) on January 6, 2005, and he was pronounced dead by Dr. Lehrach at 2129 (9:29 p.m.). CX 7 at 3-4
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reports or that he has ever personally conducted a colonoscopy. Even if he has performed colonoscopies, he clearly did not perform the colonoscopy on the Decedent, and I find that it is close to preposterous to suggest that an internist reading a report of a colonoscopy, which is not in evidence, is in a better position to determine whether it was negative for cancer than the gastroenterologist who actually examined the Decedent's colon.[23] This point is critical because Dr. Pulde stated that lung cancer is the most common type of internal cancer and that he agreed with Dr. Connors' initial assessment that Decedent likely had either colon or lung cancer as his primary cancer. EX-11 at 35-37. Third, Dr. Pulde ultimately agreed that "a primary lung cancer is a good probability" though he declined to make this finding "within a reasonable degree of medical certainty." EX 11 at 16. For these reasons, I give little weight to Dr. Pulde's altered opinion on the primary source of the Decedent' cancer and conclude on the basis of Dr. Connors' reasoned opinions that the evidence establishes that it is more likely than not that the Claimant's primary cancer was in his lung.

Causal Relationship of the Decedent's Lung Cancer to Employment

Drs. Matarese, Daum, Pulde and Harbison all addressed whether the Decedent had lung cancer and whether it was related to his employment at EB. I give little weight to Dr. Matarese's opinion because it is cursory and includes his assumption that the Decedent had asbestosis which is contrary to my finding that a preponderance of the evidence does not establish the presence of asbestosis. In light of my finding that the presence of pleural plaques in the Decedent's lungs has been established by a preponderance of the evidence, the causation issue turns on the relative persuasiveness carried by Dr. Daum's opinion that the Decedent's asbestos exposure, as confirmed by the presence of pleural plaques, in synergistic combination with his cigarette smoking significantly increased his risk of developing lung cancer in comparison to the opinions expressed by Drs. Pulde and Harbison that asbestos exposure alone, even with pleural plaques and cigarette smoking, are not associated with increased lung cancer risk in the absence of asbestosis. On this pivotal point, all three physicians rely on scientific literature dealing with the cancer risks associated with asbestos exposure. In particular, Dr. Daum cited the Helsinki Criteria as supporting her opinion that the Decedent's lung cancer risk was doubled by his asbestos exposure, and she added that cigarette smoking interacted with the asbestos exposure to multiply the Claimant's risk. The Helsinki Criteria or "Consensus Report" was produced by The International Expert Meeting on Asbestos, Asbestosis and Cancer, a multidisciplinary group of 19 participants from eight non-asbestos producing nations, which convened in Helsinki, Finland in January of 1997. 23 Scand. J. Work Environ. Health 311 (1997).[24] The Helsinki participants

[23] It should be noted that this situation is distinguishable from the cases where courts have criticized ALJs for crediting autopsy prosectors over reviewing pathologists simply because the prosector had the opportunity to view the whole body as opposed to the tissue samples examined by the other pathologists. *See, e.g., Peabody Coal Co. v. McCandless*, 255 F.3d 465, 468 (7th Cir. 2001); *Bill Branch Coal Corp. v. Sparks*, 213 F.3d 186, 191-192 (4th Cir. 2000); *Freeman United Coal Mining Co. v. Stone*, 957 F.2d 360, 362-63 (7th Cir. 1992). Here, Dr. Pulde only read a report and examined no tissue samples.

[24] The report states that the multidisciplinary group consisted of pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists, and clinical and laboratory scientists specializing in tissue fiber analysis. 23 Scand. J. Work Environ. Health at 311. There were four participants from the United States, including a representative from NIOSH and Dr. Victor Roggli whose papers from 1990 and 1994 are cited in Dr. Harbison's bibliography (EX 3 at 13). *Id.* at 315-316. Indeed, Dr. Roggli's 1990 paper, published at 88 Environ. Health Perspective 295 (1990), was cited by Dr. Harbison for the proposition that patients with

stated that since all types of lung cancer can be related to asbestos, the "histological type of a lung cancer and its anatomic location (central or peripheral, upper lobe versus lower lobe) are of no significant value in deciding whether or not an individual lung cancer is attributable to asbestos." *Id.* at 313. Regarding the relative risk of developing lung cancer, the Consensus Report in pertinent part states,

> As examples, 1 year of heavy exposure (eg, manufacture of asbestos products, asbestos spraying, insulation work with asbestos materials, demolition of old buildings) or 5–10 years of moderate exposure (eg, construction, shipbuilding) may increase the lung cancer risk 2-fold or more. In some circumstances of extremely high asbestos exposure, a 2-fold risk of lung cancer can be achieved with exposure of less than 1 year.
>
> The relative risk of lung cancer is estimated to increase 0.5–4% for each fiber per cubic centimeter per year (fiber-years) of cumulative exposure. With the use of the upper boundary of this range, a cumulative exposure of 25 fiber-years is estimated to increase the risk of lung cancer 2-fold. Clinical cases of asbestosis may occur at comparable cumulative exposures.
>
> * * * * *
>
> Estimates of the relative risk for asbestos-associated lung cancer are based on different-sized populations. Because of the high incidence of lung cancer in the general population, it is not possible to prove in precise deterministic terms that asbestos is the causative factor for an individual patient, even when asbestosis is present. However, attribution of causation requires reasonable medical certainty on a probability basis that the agent (asbestos) has caused or contributed materially to the disease. The likelihood that asbestos exposure has made a substantial contribution increases when the exposure increases. Cumulative exposure, on a probability basis, should thus be considered the main criterion for the attribution of a substantial contribution by asbestos to lung cancer risk. For example, relative risk is roughly doubled for cohorts exposed to asbestos fibers at a cumulative exposure of 25 fiber-years or with an equivalent occupational history, at which level asbestosis may or may not be present or detectable. Heavy exposure, in the absence of radiologically diagnosed asbestosis, is sufficient to increase the risk of lung cancer. Cumulative exposures below 25 fiber-years are also associated with an increased risk of lung cancer, but to a less extent.
>
> The presence of asbestosis is an indicator of high exposure. Asbestosis may also contribute some additional risk of lung cancer beyond that conferred by asbestos exposure alone. Asbestosis diagnosed clinically, radiologically (including HRCT), or histologically can be used to attribute a substantial causal or contributory role to asbestos for an associated lung cancer.

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asbestosis have a marked risk of lung cancer while "the risk of lung cancer attributable to asbestos in exposed workers who also smoke is controversial." EX 3 at 11.

> Pleural plaques are an indicator of exposure to asbestos fibers. Because pleural plaques may be associated with low levels of asbestos exposure, the attribution of lung cancer to asbestos exposure must be supported by an occupational history of substantial asbestos exposure or measures of asbestos fiber burden. Bilateral diffuse pleural thickening is often associated with moderate or heavy exposures, as seen in cases with asbestosis, and should be considered accordingly in terms of attribution.
>
> A minimum lag-time of 10 years from the first asbestos exposure is required to attribute the lung cancer to asbestos.
>
> Not all exposure criteria need to be fulfilled for the purposes of attribution. For example, the following can be considered: (i) significant occupational exposure history with low fiber burdens (eg, long exposure to chrysotile and long lag-time between the end of exposure and mineralogical analysis) and (ii) high fiber counts in lung or broncholavage fluid with an uncertain history or without long-term duration (short exposures can be very intense).
>
> At very low levels of asbestos exposure, the risk of lung cancer appears to be undetectably low.
>
> Although tobacco smoking affects the total lung cancer risk, this effect does not detract from the risk of lung cancer attributable to asbestos exposure. No attempt has been made in this report to apportion the relative contributions of asbestos exposure and tobacco smoking.

Id. at 313-314. Dr. Pulde stated that he respects Dr. Daum's opinion, but he cited several studies, none of which are in evidence, as indicating that the aggregate of scientific research does not support an increased risk of lung cancer from asbestos exposure in the absence of asbestosis. EX 11 at 24-25. Dr. Harbison similarly cited scientific authority which questions whether asbestos exposure without asbestosis in a smoker increases the risk of lung cancer, and he also asserted that the Claimant had additional risk factors for lung cancer which had not been properly ruled out. EX 3 at 11-12. Dr. Daum did not discuss the studies cited by Drs. Pulde and Harbison, and neither Dr. Pulde nor Dr. Harbison mentioned the Helsinki Criteria.

The divergent opinions of the medical experts in this case indicate that the relationship between asbestos exposure and lung cancer remains somewhat controversial, at least in the absence of parenchymal asbestosis. In the instant case, I am persuaded after consideration of the entire body of evidence contained in the record that that Dr. Daum's opinions, while not dispositive of all doubt in the matter, are generally entitled to greater weight than the contrary views expressed by Drs. Pulde and Harbison. Specifically, I credit Dr. Daum's opinion that the Decedent's asbestos exposure at EB exceeded the 25 fiber-year threshold at which the relative risk of lung cancer is doubled according to the Helsinki Criteria and below which, according to Dr. Pulde, an asbestos-related lung condition is unlikely. Granted, the record contains no air quality test results or other data that would permit a precise estimation of the Decedent's cumulative asbestos cumulative exposure or dose. However, Dr. Daum studied asbestos

exposure among workers at EB's Groton shipyard during the time that the Decedent worked there as a painter/cleaner, and her assumptions regarding the Decedent's asbestos exposure are substantially more consistent with the credible testimony of E.T. and the 1986 report of Dr. Cherniack (noting consistent exposure to asbestos dust from 1963 to at least the mid-1970s) than Dr. Pulde's assumption that his exposure was no more than "infrequent and indirect."[25] Therefore, I find that a preponderance of the evidence establishes that the Decedent had at least moderate exposure, as that term is used in the Helsinki Criteria, to asbestos through his work at EB as a painter/cleaner between 1963 and the mid-1979s, resulting in a cumulative exposure of at least 25 fiber-years.

Having credited Dr. Daum's estimate that the Decedent has at least 25 fiber-years of asbestos exposure, the next question is whether her opinion that the Decedent's asbestos exposure played a contributory role in the development of his lung cancer outweighs the opposing opinions from Drs. Pulde and Harbison. In this regard, I recognize that there is some controversy in the medical and scientific communities as to whether asbestos exposure in the absence of asbestosis increases the relative risk of developing a lung cancer. However, in weighing the relative merits of the competing viewpoints on this question, I find it reasonable to defer to the Helsinki Criteria's determination that "5–10 years of moderate exposure (eg, construction, shipbuilding) may increase the lung cancer risk 2-fold or more" and that "relative risk is roughly doubled for cohorts exposed to asbestos fibers at a cumulative exposure of 25 fiber-years or with an equivalent occupational history, at which level asbestosis may or may not be present or detectable." CX 12 (Claimant's Exhibit A); 23 Scand. J. Work Environ. Health at 313-314 (underlining supplied). In my view, the multi-national and multidisciplinary composition of the Helsinki participants makes it more likely that their findings and opinions represent the current medical and scientific consensus on the causal relationship between asbestos exposure and lung cancer than the individual studies cited by Drs. Pulde and Harbison. Therefore, I credit Dr. Daum's opinion that the Decedent's asbestos exposure was sufficient to double his risk of lung cancer.

At the same time, I do not credit Dr. Daum's opinions that the Decedent's lung cancer risk was further multiplied by his cigarette smoking history and/or by his other exposure to carcinogens such as chromium paints at EB. The Helsinki Criteria does not address whether there is an interactive, synergistic or multiplicative relationship between asbestos and cigarette smoking in terms of relative lung cancer risk,[26] and Dr. Daum cited no specific scientific authority which rebuts Dr. Pulde's assertion that the medical literature shows no synergistic

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[25] Dr. Daum testified that the Decedent had at least 25 fiber-years of asbestos exposure before she was fed the previously-noted miscalculations by the Claimant's attorney in his attempt to quantify the Decedent's exposure. Therefore, I find that her opinion that the Decedent had at least 25 fiber-years of asbestos exposure is not tainted by counsel's mathematical errors.

[26] Rather, the Helsinki criteria simply states,

> Although tobacco smoking affects the total lung cancer risk, this effect does not detract from the risk of lung cancer attributable to asbestos exposure. No attempt has been made in this report to apportion the relative contributions of asbestos exposure and tobacco smoking.

CX 12 (Claimant's Exhibit A); 23 Scand. J. Work Environ. Health at 314.

relationship in the absence of asbestosis. At best, the evidence of record on this issue is inconclusive. The record is also inconclusive on the role played by the Decedent's occupational exposure to chromium and any other unspecified workplace carcinogen. While there is lay evidence that the Decedent worked with paints containing chromium, which Dr. Daum identified as a carcinogen that likely contributed to the Decedent's cancer, there is no reliable evidence regarding the extent of his exposure and whether such exposure was sufficient to cause harm. Therefore, I conclude that a preponderance of the evidence fails to establish that the Decedent's relative risk of lung cancer from asbestos exposure was further increased by either his cigarette smoking or his occupational exposure to chromium or any other carcinogen at EB.

Dr. Daum's credited opinion that the Decedent's asbestos exposure alone at least doubled his risk of lung cancer satisfies the "greater than 2.0" relative risk threshold at which an inference of specific causation can be drawn. *See* Federal Judicial Center, *Reference Guide on Scientific Evidence, Second Ed.* (2004) at 384. *See also In re Joint Eastern & Southern District Asbestos Litigation (Maiorana v. United States Mineral Products Co.)*, 52 F.3d 1124, 1128 (2d Cir. 1995). Therefore, I conclude that the Claimant has satisfied her burden of proving by a preponderance of the evidence that the Decedent's exposure to asbestos in the course of his employment at EB contributed to his development of lung cancer.[27] Since all the doctors agree that lung cancer caused the Decedent's death, I further conclude that the Claimant has successfully proved by a preponderance of the evidence that a work-related injury, lung cancer, caused the Decedent's death.

C. What is the applicable average weekly wage?

Average weekly wage ("AWW") determinations which form the basis of compensation rates are governed by section 10 of the Act which is designed to establish an injured worker's earning capacity at the time of the injury. *Johnson v. Newport News Shipbuilding & Dry Dock Co.*, 25 BRBS 340, 343-344 (1992). Judge Di Nardi awarded the Decedent compensation based on an AWW of $822.40, which was calculated from his wages during his final year of employment at EB, because he was diagnosed with an occupational disease within one year of his voluntary retirement from EB. ALJ Di Nardi Decision and Order at 16. Judge Di Nardi's finding was based on section 10(d)(2) of the Act which provides:

[27] It is noted that Dr. Harbison identified other non-occupational risk factors which, if not ruled out or minimized, would preclude a finding that the Claimant has met her burden of proving by a preponderance of the evidence that asbestos exposure caused, contributed to or aggravated the Decedent's lung cancer,. *See Cavallo v. Star Enterprise*, 892 F. Supp. 756, 771 (E.D. Va. 1995) ("[i]f other possible causes of an injury cannot be ruled out, or at least the probability of their contribution to causation minimized, then the 'more likely than not' threshold for proving causation may not be met."), *aff'd on this ground, rev'd on other grounds*, 100 F.3d 1150 (4th Cir. 1996), *cert. denied*, 522 U.S. 1044 (1998); *Ruggiero v. Warner-Lambert Co.*, 424 F.3d 249, 254 (2d Cir. 2005). The non-occupational risk factors cited by Dr Harbison are "[a]ge, body weight, physical activity, and diet." EX 3 at 6. However, he simply mentioned these factors and provided no discussion of the extent to which they increased the Decedent's lung cancer risk and no credible explanation that the diet and physical activity factors are even applicable in the Decedent's case. Accordingly, I find that the possible co-contribution from these potential non-occupational factor have not been shown the more than minimal and, therefore, do not appreciably reduce the weight of evidence on the Claimant's side of the balance.

> Notwithstanding paragraph (1), with respect to any claim based on a death or disability due to an occupational disease for which the time of injury (as determined under subsection (i)) occurs-
>
> (A) within the first year after the employee has retired, the average weekly wages shall be one fifty-second part of his average annual earnings during the 52-week period preceding retirement; or
>
> (B) more than one year after the employee has retired, the average weekly wage shall be deemed to be the national average weekly wage (as determined by the Secretary pursuant to section 6(b)) applicable at the time of the injury.

33 U.S.C. § 10(d)(2). The Claimant contends that if the Decedent's work-related lung disease is found to have hastened his death, section 10(d)(2)(A) would apply, and benefits should be awarded based on the AWW of $822.40. Claimant Br. at 18. Alternatively, the Claimant suggests that if the Decedent's lung cancer is determined to be the sole cause of death, section 10(d)(2)(B) would require that the National Average Weekly Wage ("NAWW") of $523.58 be utilized. *Id.*[28] In the event that both the underlying work-related lung disease and lung cancer are found to have contributed to the Decedent's death, the Claimant states that there would be two injuries with two different benefit rates, but she argues that the $822.40 AWW should prevail over the $523.58 NAWW in order to prevent EB from benefiting from the fact that its workplace caused the Decedent to suffer a second injury. *Id.* at 18-19. EB counters that if benefits are awarded, the NAWW in effect on December 29, 2004 when the Decedent was diagnosed with lung cancer would govern. EB Br. at 21. Neither party has cited any authority for its position on the applicable AWW.

Section 9(b) of the Act provides for payment of widow's compensation equal to "50 per centum of the average wages of the deceased . . . ." 33 U.S.C. § 909(b). The Decedent's average weekly wage, as determined by Judge Di Nardi in the prior proceeding pursuant to section 10(d)(2)(A), was $822.40. While the Act requires use of the NAWW in lieu of a decedent's AWW for computation of death benefits in cases where the decedent's AWW is less than the NAWW; 33 U.S.C. § 909(e);[29] there is nothing in the Act or the case law that would permit use

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[28] $523.58 is the NAWW in effect for the period of 10/1/04 to 9/30/05 when the Decedent's lung cancer became manifest. *See* http://www.dol.gov/esa/owcp/dlhwc/NAWWinfo.htm.

[29] Section 9(e) states,

> In computing death benefits, the average weekly wages of the deceased shall not be less than the national average weekly wage as prescribed in section 6(b), but --
>
> (1) the total weekly benefits shall not exceed the lesser of the average weekly wages of the deceased or the benefit which the deceased employee would have been eligible to receive under section 6(b)(1); and
>
> (2) in the case of a claim based on death due to an occupational disease for which the time of injury (as determined under section 10(i) occurs after the employee has retired, the total weekly benefits shall not exceed one fifty-second part of the employee's average annual earnings during the 52-week period preceding retirement.

of the NAWW when it is less than a decedent's AWW.[30] Indeed, to apply the NAWW in this case where the Decedent was receiving compensation based his AWW and thus award the Claimant compensation at a substantially lower rate because the Decedent had the misfortune to develop lung cancer in addition to his COPD would fly in the face of the well-established admonition that the Act must be liberally construed in order to effectuate its humanitarian and remedial purposes and to avoid "harsh and incongruous results." *Voris v. Eikel*, 346 U.S. 328, 333 (1953). *See also Director, OWCP v. Newport News Shipbuilding & Dry Dock Co*., 514 U.S. 122, 135-136 (1995). Therefore, I conclude that the Decedent's average wages for computation of the Claimant's benefits pursuant to section 9(b) were $822.40.

D. The Claim for Increased Disability Compensation

As set forth above, the Claimant in her brief has requested permanent total disability compensation of behalf of the Decedent's estate for the period of December 29, 2004 to January 6, 2005. Claimant Br. at 19. Though the amount of compensation at stake is minimal since the Decedent was paid compensation for this period pursuant to Judge Di Nardi's order, the issue of entitlement to disability compensation cannot be addressed without EB first being provided with notice and an opportunity to offer responsive evidence and argument. 20 C.F.R. § 702.336(b); *Estate of Cowart v. Nicklos Drilling Co*., 23 BRBS 42, 47-48 (1989), *rev'd in part*, 907 F.2d 1552 (5th Cir. 1990), *aff'd en banc*, 927 F.2d 828 (1991), *aff'd*, 505 U.S. 469 (1992); *Cornell Univ. v. Velez*, 856 F.2d 402, 405 (1st Cir. 1988). Since it is unlikely that the claim for additional disability compensation will be controversial once a final determination is made with respect to the cause of the Decedent's death, I will not reopen the record at this point and further delay adjudication of the death claim. In the event that any dispute between the parties over the disability compensation remains after a final determination is made in the death claim, the Decedent's estate may pursue the matter under section 22 of the Act as a petition for

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33 U.S.C. § 909(e). *See also Donovan v Newport News Shipbuilding and Dry Dock Co*., 31 BRBS 2, 3-5 (1997) (affirming award of widow's compensation at 50 percent of the NAWW plus annual adjustments pursuant to section 10(f) as not inconsistent with the compensation cap imposed by section 9(e)(1) even though the section 10(f) adjustments pushed the widow's compensation rate above the decedent's AWW). In *Donovan*, the decedent had been awarded disability compensation commencing in 1978 for occupational lead poisoning based on his AWW of $228.33. 31 BRBS at 3. After he died in 1995 from work-related poisoning, his widow was awarded compensation pursuant to sections 9(b) and (e) based on 50 percent of the NAWW which was $380.46. *Id.*

[30] There is a substantial body of case law addressing when an occupational disease such as lung cancer is deemed to have occurred for purposes other than determining the applicable AWW. *See, e.g., Insurance Co. of North America v. U.S. Dept. of Labor*, 969 F.2d 1400, 1403-1406 (2d Cir. 1992), *cert. denied*, 507 U.S. 909 (1993) (date of manifestation of occupational disease with long latency such as asbestos-related lung cancer determines whether the 1972 situs amendments to the Act are applicable); *Travelers Insurance Co. v. Cardillo*, 225 F.2d 137, 142-143 (2d Cir. 1955), *cert. denied*, 350 U.S. 913 (1955) (Act's statute of limitations does not begin to run until occupational disease becomes manifest). Since these cases do not address AWW in a death benefits scenario, I find that they are not controlling. A more analogous case is *Bath Iron Works Corp. v. Director, U.S. Dept. of Labor*, 193 F.3d 27, 32 (1st Cir. 1999) where the Court held that an initial asbestos-related injury was aggravated by further exposure to pulmonary irritants, causing a "new" injury and resulting in an increase in benefits payable by a new carrier and based upon the AWW at the time of the new injury). In this case, since there was no additional occupational exposure to injurious stimuli, there is no basis for a finding of a new injury and calculating a different AWW.

modification of Judge Di Nardi's compensation order. Accordingly, no findings are made herein with respect to the Decedent's entitlement to additional disability compensation or the merits of any section 22 petition for modification.

E. Summary of Benefits Awarded

1.Death and Survivor's Benefits

As a surviving spouse who was married to and living with the Decedent at the time of his work-related death, the Claimant is entitled to the death benefits and funeral expenses provided by section 9 of the LHWCA. *See Griffin v. Bath Iron Works Corp.*, 25 BRBS 26, 29 (1991). The Claimant introduced receipts showing that she paid a total of $6,250.67 for the Decedent's funeral. CX 3. Pursuant to section 9(a) of the LHWCA, which allows for funeral expense reimbursement up to a maximum of $3,000.00, I find that she is entitled to an award of $3,000.00 in funeral expenses. I further conclude that the Claimant is entitled to survivor's compensation pursuant to section 9(b) of the LHWCA at the rate of 50 percent of the Decedent's average weekly wages of $822.40, which produces a base compensation rate of $411.20 per week. This base compensation rate is subject to the annual increases provided for by section 10(f) of the LHWCA. *Donovan v Newport News Shipbuilding and Dry Dock Co*., 31 BRBS 2, 3-5 (1997).

2. Interest

Interest is due on all unpaid compensation including funeral expenses. *Adams v. Newport News Shipbuilding & Dry Dock Co*., 22 BRBS 78, 84 (1989). The appropriate interest rate shall be determined pursuant to 28 U.S.C. § 1961 (2003) as of the filing date of this Decision and Order with the District Director. My order incorporates by reference this statute and provides for its specific administrative application by the District Director. The appropriate rate shall be determined as of the filing date of this Decision and Order with the District Director.

3. Medical Expenses

As discussed above, EB is liable pursuant to section 7(a) of the LHWCA for those medical expenses reasonably and necessarily incurred as a result of a work-related injury. *Colburn v. General Dynamics Corp.*, 21 BRBS 219, 222 (1988); *Parnell v. Capitol Hill Masonry*, 11 BRBS 532, 539 (1979). In addition, EB will be ordered to reimburse the Decedent's estate for any payments already made for medical bills reasonably and necessarily incurred in connection with the Decedent's work-related lung cancer.

4. Attorney's Fees

Having successfully established her right to compensation and medical benefits through the services of an attorney, the Claimant is entitled to an award of attorney's fees under section 28 of the LHWCA. *American Stevedores v. Salzano*, 538 F.2d 933, 937 (2nd Cir. 1976). Her attorney will be granted leave to submit an application for fees and

costs in accordance with the requirements of 20 C.F.R. § 702.132, and EB will be allowed 15 days from service of the fee application to file any objection to the requested fees and expenses.

V. Order

Based upon the foregoing Findings of Fact and Conclusions of Law and upon the entire record, the following compensation order is entered:

(1) Electric Boat Corporation shall pay to the Widow survivor's compensation pursuant to 33 U.S.C. § 909(b) at the base rate of $411.20 per week, with the applicable annual adjustments provided in 33 U.S.C. § 910(f), commencing April 3, 2005 and continuing until death or remarriage, plus interest on all past due compensation at the Treasury Bill rate applicable under 28 U.S.C. § 1961 (2003), computed from the date each payment was originally due until paid;

(2) Electric Boat Corporation shall pay to the Widow funeral expenses in the statutory maximum amount of $3,000.00, plus interest on all such expenses at the Treasury Bill rate applicable under 28 U.S.C. § 1961 (2003), computed from the date each expense was originally due until paid;

(3) Electric Boat Corporation is responsible for reasonable and necessary medical expenses incurred by the Decedent for treatment of his work-related COPD and lung cancer, and Electric Boat Corporation shall reimburse the Decedent's estate for any payments already made for medical bills reasonably and necessarily incurred in connection with the Decedent's work-related lung disease;

(4) The Claimant's attorney shall have 30 days from the date this decision and order is filed with the District Director to file any objection to the fees and expenses requested by the Claimant's attorney, and Electric Boat Corporation shall have 15 days from service of the fee application to file any objection; and

(5) All computations of benefits and other calculations provided for in this Order are subject to verification and adjustment by the District Director.

SO ORDERED.

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DANIEL F. SUTTON
Administrative Law Judge

Boston, Massachusetts